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The effect of maternal obesity on the course of labor

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Abstract

Aim: To determine whether maternal obesity is associated with dysfunctional labor patterns.
Methods: In a case-control design we compared the graphic labor patterns of a group of 105 very obese subjects [body mass index (BMI) >35 kg/m^2] with those of 113 lean controls (BMI <26 kg/m^2). All entered spontaneous labor at term. Cases with birth weights >4 kg, diabetes mellitus, hypertension and prior cesarean delivery were excluded.
Results: The obese group had a significantly higher frequency of arrest of dilatation (17.6 vs. 5.2%; $P=0.005$).
Conclusions: Maternal obesity is associated with active phase labor dysfunction, specifically arrest of dilatation.

Keywords: Body mass index (BMI); dysfunctional labor; labor; maternal obesity.

Introduction

The current epidemic of obesity in the developed world has adversely affected public health in many ways. It has contributed to the prevalence and severity of medical conditions including diabetes mellitus, hypertension, heart disease, and certain types of cancer [20]. It has also created reproductive adversity. Many obesity-related illnesses complicate pregnancies. Moreover, maternal obesity is associated with excess fetal growth, which increases the likelihood of cesarean section and of birth injury, as well as potential life-long health risks [11].

Many studies have demonstrated a strong relationship between maternal obesity and the risk of cesarean delivery [1, 5, 7, 10, 13–17, 19, 23]. The reasons for this high cesarean rate are, however, not entirely clear. It relates in part to the association of obesity with diabetes and hypertension [14], which themselves predispose to the need for cesarean. In addition, the high prevalence of fetal macrosomia [1, 9, 16, 23, 26] among obese women probably leads to more cesareans for cephalopelvic disproportion. These factors do not, however, explain completely the high obesity-related cesarean rate noted in most studies. High cesarean rates have been found in otherwise low-risk obese women [8, 26], and multivariate analyses support obesity as an independent risk factor associated with cesarean delivery [17, 18]. A possible explanation is that obesity might somehow create dysfunctional labor, independent of the presence of true fetopelvic disproportion.

To enhance our understanding of the association between maternal obesity and labor progress, we studied graphic labor patterns in a group of very obese women and compared them to those of lean controls. We hypothesized that maternal obesity affects the likelihood of an arrest of dilatation, and other objectifiable labor abnormalities.

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Methods

This retrospective case-control study qualified for expedited review from Jamaica Hospital Medical Center's Institutional Review Board. Our goal was to compare the labors of a group of very obese women [body mass index (BMI) >35 kg/m^2] with those of a control group with a normal BMI (<26 kg/m^2). Both groups would consist of term singleton pregnancies without neonatal macrosomia, diabetes, or other complications. These exclusions eliminated most of the factors associated with obesity known to increase the risk of dysfunctional labor.

We estimated a sample size of 200 would be necessary to have a power of 0.80 to detect a three-fold increase in the frequency of arrest of dilatation with an alpha of 0.05. Based on our hospital's known prevalence of macrosomia, diabetes mellitus, multiple gestations and preterm deliveries, we estimated that about 400 cases would be necessary to provide 200 cases for analysis after exclusions.

To constitute the groups of cases and controls, we used our electronic medical record (E&C Medical Intelligence Inc., New York, NY) to identify the first 200 consecutive obstetric cases with a maternal BMI >35 kg/m^2 and 200 consecutive normal weight cases (BMI <26 kg/m^2) beginning in October 2003. From these, we excluded multiple gestations, cases with birth weight >4000 g or gestational age <37 completed weeks, intrauterine growth restriction, diabetes mellitus, severe hypertension or preeclampsia, and major fetal anomalies. We also excluded all inductions of labor, patients with a prior cesarean, and cases delivered by cesarean prior to labor. Of the remaining 254 cases

we eliminated 36 in which the records had insufficient data about labor progress to allow graphic analysis. Remaining after these exclusions were 105 obese and 113 lean cases. Thus, the case and control groups consisted of uncomplicated term pregnancies with spontaneous labors. The cases were very obese and the controls lean. The goal of this selection process was to isolate the effects of obesity *per se* on the course of labor.

Pertinent data regarding labor and delivery were abstracted from the medical records of all cases. Examinations of cervical dilatation and fetal station were plotted on a graph by one of the investigators, blinded to whether the patient was lean or obese. The resulting curves of cervical dilatation and fetal descent were analyzed, and labor dysfunctions classified according to the method of Friedman [6]. Labor dysfunctions are defined in Appendix 1. Data were entered into a statistical software system (JMP, version 5.0; SAS Statistical Software, Cary, NC) for analysis. Differences between the two groups were sought with χ^2 testing for categorical variables and Student's *t*-test for continuous variables. In instances in which expected cell numbers for categorical data were <5, Fisher's exact test was used. For continuous data that were not normally distributed (such as quantifiable labor variables) the non-parametric Mann-Whitney test was used.

Results

The characteristics of the two groups are summarized in Table 1. The obese patients were significantly older by about 2 years than the lean ones. The proportion of nulliparas, the cesarean rate (for all indications), and the use of oxytocin were similar in both groups. There were no operative deliveries. While the mean birth weight in the obese group was significantly larger than in the lean group ($P=0.012$) the mean difference was only 116 g, and the proportion of larger babies (3700–3999 g) was not significantly different between the two groups.

The quantifiable characteristics of the labors in the lean and obese cases were generally similar (Table 2). Exceptions were a significantly shorter latent phase, which accounted for the shorter first stage of labor among obese nulliparas.

When labor curves were analyzed for dysfunctional labor patterns, however, significant differences were found between the two groups (Table 3). While obese patients had a lower frequency of prolonged latent phase, they had a dramatically greater incidence of arrest of dilatation (17.6 vs. 5.2%, $P=0.005$). In addition, there were more patients in the obese group with protracted active phase (14.8 vs. 9.3%, $P=0.177$) and protracted descent (3.3 vs. 0%, $P=0.180$), although these differences did not reach statistical significance.

Discussion

We found a significantly higher rate of arrest of dilatation in obese when compared to lean women. We considered that the greater birth weight in the obese cases might have contributed to the higher rate of arrest disorders, even though the difference of only 116 g seems unlikely to have had a major impact on the probability of cephalopelvic disproportion. The proportion of larger babies (3700–3999 g) was not different between the groups.

Table 1 Characteristics of the sample.

	Lean (n=113)	Obese (n=105)	P-value
Body mass index (kg/m^2)	24.0±1.6	39.7±4.8	<0.001
Maternal age (years)	24.4±5.5	26.3±6.5	0.020
Nulliparity (%)	60	51	0.190
Cesarean rate (%)	8.0	10.5	0.640
Oxytocin use (%)	35	30	0.340
Birth weight (g)	3148±353	3264±321	0.012
Birth weight 3700–3999 g, n (%)	7 (6.0)	9 (9.0)	0.680

Table 2 Quantifiable labor characteristics*.

	Lean (n=113)		Obese (n=105)	
	Nulliparas	Multiparas	Nulliparas	Multiparas
Latent phase (min)	735 (333, 1617)	330 (156, 1093)	540 (153, 1062)**	330 (153, 1068)
Active phase (min)	177 (95, 394)	107 (59, 270)	143 (68, 471)	90 (48, 198)
Stage I (min)	937 (484, 1801)	487 (264, 1094)	665 (218, 1198)**	359 (164, 1225)
Stage II (min)	30 (8, 111)	14 (4, 40)	32 (11, 139)	10 (2, 55)
Deceleration phase (min)	60 (25, 148)	30 (9, 124)	50 (10, 164)	28 (10, 84)
Slope dilatation (cm/h)	2.9 (0.9, 6.1)	4.2 (2.0, 10.0)	3.5 (0.9, 7.0)	4.8 (1.8, 12.2)
Slope descent (cm/h)	6.2 (2.7, 17.2)	15.5 (5.1, 44.5)	6.0 (1.6, 24.4)	17.8 (4.8, 49.4)

*All values are median (10th, 90th percentile).
**Significantly different from lean group of same parity.

Table 3 Dysfunctional labor patterns.

	Lean	Obese	P-value
Prolonged latent phase n/total* (%)	12/65 (18.5)	5/74 (6.8)	<0.001
Protracted active phase n/total (%)	9/97 (9.3)	13/88 (14.8)	0.177
Arrest of dilatation n/total (%)	5/96 (5.2)	16/91 (17.6)	0.005
Prolonged deceleration phase n/total (%)	12/89 (13.5)	9/90 (10.0)	0.180
Failure of descent n/total (%)	1/102 (1.0)	0/92 (0)	0.526
Protracted descent n/total (%)	0/100 (0)	3/89 (3.3)	0.108
Arrest of descent n/total (%)	5/103 (4.9)	1/91 (1.1)	0.137

*Total cases in each cell vary because in some cases an insufficient number of dilatation or descent measurements were documented in the medical record to allow definitive diagnosis.

Moreover, among cases with birth weights below 3500 g, in which disproportion would be uncommon, there were 5.0% of lean women who had arrest of dilatation, compared with 18.8% of the obese cases ($P=0.002$). The birth weights among the cases with arrest of dilatation were slightly, but not significantly, lower than those of the lean controls with arrest disorders (3396 ± 273 vs. 3280 ± 265 g; $P=0.399$). We conclude that the nearly three-fold higher frequency of arrest of dilatation was not explainable by the small difference in birth weight between the two groups.

Maternal age is another potential confounder, because older women are more likely to have dysfunctional labor patterns [3], but the difference of only 2 years in the mean age of the groups is unlikely to explain the observed difference in dysfunctional labor. Moreover, in subjects with arrest of dilatation, we found no difference in maternal age. In fact, the lean cases with arrest were slightly older (25.4 vs. 24.6 years; $P=0.804$). Thus, it is improbable that the observed difference in arrest of dilatation observed in the obese patients is due to higher birth weights or older maternal age among them.

Although there were more protracted active phase disorders in the obese group (14.8 vs. 9.3%) this difference was not statistically significant. The difference could have been muted by the fact that there were more nulliparas in the lean group, and nulliparas would be expected to have substantially more protraction disorders than multiparas [6]. The failure to find a significant difference could also have been a type II error, in that a sample size of >500 cases would have been necessary to have 80% power to detect a doubling in the frequency of protracted active phase.

There was no significant difference in the frequency of disorders of the pelvic division of labor (prolonged deceleration phase, failed, arrested or protracted descent), suggesting that the observed effects of obesity were confined to the first stage, and did not influence abnormalities of descent. Second stage abnormalities are more closely linked to bony disproportion than are active phase disorders, which more often are associated with deficient contractility. If obesity impairs contractility, it makes sense that its effects would be observed primarily in the first stage.

Consistent with this notion are the observations of Buhimschi et al., who showed no difference in intrauterine pressures generated during the second stage among groups of obese, overweight, and normal weight women [2].

Our finding of fewer latent phase disorders in obese nulliparas seems inconsistent with the hypothesis that obesity hinders contractility. However, the actual median difference in latent phase duration was small (about 2 h) and may not be clinically meaningful. The length of the latent phase is especially difficult to ascertain from this kind of retrospective analysis, because of the unavoidable subjectivity in identifying the onset of labor. By contrast, active phase slopes and patterns of dilatation are readily determinable from most records.

Other clinical evidence is in harmony with the potential for obesity to compromise the intensity or efficiency of uterine contractility. Onset of labor may be delayed by obesity, and dysfunctional labor may be abetted. Obese women have been observed to have longer-term gestations, and more post-dates pregnancies than thinner women [4, 22]. Also, in a prior analysis of labor duration, Vahratian et al. studied rates of dilatation in a data base of 612 nulliparas and found active phase labor (defined as 4–10 cm dilatation) was significantly longer in overweight and obese women, after adjusting for birth weight [24]. Zhang et al. [27] showed in a clinical study that obese women had a higher risk of cesarean, related primarily to abnormal progress in the first stage of labor.

Until recently, there has been no biologic basis to explain why simply being obese would affect labor, but accumulating evidence supports the association of obesity with impaired uterine contractility. In one study, myometrial tissue obtained at cesarean from obese

women was shown *in vitro* to contract with less force (as indicated by lower calcium fluxes) than those from normal weight subjects [27].

The basis for this contractile inhibition may reside in some of the biochemical changes induced by obesity. For example, leptin, a protein with diverse metabolic and regulatory functions, is produced in increased amounts in obese individuals. Moynihan et al. demonstrated that leptin strongly inhibited myometrial contractility *in vitro* [12]. Cholesterol, also increased in obesity, has similar inhibitory effects on myometrial activity and calcium signaling [21, 28].

We conclude that obesity may interfere with the progress of labor, resulting in dysfunctional patterns of dilatation. The mechanism for such an effect may be mediated through diminished uterine contractility in the active phase of labor, a consequence of increased levels of leptin, cholesterol, or other metabolic features of the obese state. Obesity should be added to the list of possible causes of abnormal labor progression.

The implications of our findings are considerable because of the rising prevalence of obesity in the pregnant population. Obstetricians should be aware that dysfunctional labor patterns in the first stage may be attributable to compromised contractility in these patients, and not necessarily a consequence of disproportion. The fact, however, that obesity does predispose to fetal macrosomia can make this a challenging clinical decision. Whether the presumed effects of obesity on uterine contractility can be readily overcome by the use of oxytocin is unknown and requires study.

Despite the convincing difference we found in the frequency of arrest of dilatation, our study was not sufficiently powered to demonstrate with confidence differences in other dysfunctional labor patterns. The sample, however, had the advantage over large data bases in that it was subject to meticulous review of the clinical details of every record, so we are confident of the accuracy of the labor diagnoses and other clinical information. Unfortunately, no meaningful information about uterine activity could be gleaned from the records, because intrauterine pressure catheters were used infrequently in the study institution. Therefore, we can only speculate about the mechanism that underlies our findings.

In summary, we demonstrated a more than three-fold higher rate of arrest of dilatation among very obese women compared to those who were lean. Interference with normal progress of labor may therefore be among the many adverse health consequences of marked obesity. The mechanism underlying this observation is not known, but may be related to the effects of obesity on uterine contractility.

Appendix 1. Definitions of labor terms [6, 25]

Labor curve: A plot of the relationships among cervical dilatation, fetal station, and elapsed time in labor.

Latent phase: Portion of labor from onset until the acceleration in cervical dilatation seen at onset of active phase.

Active phase: Portion of labor from end of latent phase to full cervical dilatation.

Deceleration phase: The terminal portion of active phase dilatation when the cervix is approaching the widest diameter of the presenting part. Generally this occurs between about 8 cm and full dilatation.

Prolonged latent phase: Exceeds 20 h in nulliparas or 14 h in multiparas.

Protracted active phase: Linear dilatation in active phase <1.2 cm/h in nulliparas or <1.5 cm/h in multiparas.

Arrest of dilatation: No progress in dilatation for 2 h in active phase labor.

Prolonged deceleration phase: Exceeds 3 h in nulliparas or 1 h in multiparas.

Failure of descent: No descent of the head from early labor to beyond the onset of deceleration phase or second stage.

Protracted descent: Head descent in second stage <1 cm/h in nulliparas or 2 cm/h in multiparas.

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